Thermoregulation during Exercise in the Heat : Strategies for Maintaining Health and Performance

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Thermoregulation during Exercise in the Heat

Strategies for Maintaining Health and Performance

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Abstract

As a result of the inefficiency of metabolic transfer, >75% of the energy that is generated by skeletal muscle substrate oxidation is liberated as heat. During exercise, several powerful physiological mechanisms of heat loss are activated to prevent an excessive rise in body core temperature. However, a hot and humid environment can significantly add to the challenge that physical exercise imposes on the human thermoregulatory system, as heat exchange between body and environment is substantially impaired under these conditions. This can lead to serious performance decrements and an increased risk of developing heat illness. Fortunately, there are a number of strategies that athletes can use to prevent and/or reduce the dangers that are associated with exercise in the heat. In this regard, heat acclimatisation and nutritional intervention seem to be most effective. During heat

acclimatisation, the temperature thresholds for both cutaneous vasodilation and the onset of sweating are lowered, which, in combination with plasma volume expansion, improve cardiovascular stability. Effective nutritional interventions include the optimisation of hydration status by the use of fluid replacement beverages. The latter should contain moderate amounts of glucose and sodium, which improve both water absorption and retention.

It has been well established that exercise in the heat, especially if there is high humidity, can adversely affect performance and may even result in serious heat illness, such as heat exhaustion and heat stroke. Nevertheless, major championship events are often scheduled for the hottest part of the day during the summer. This article focuses on the problems associated with exercise in the heat and the strategies that athletes can use to minimise the impact of environmental conditions on performance. To provide the reader with a basic understanding of human thermoregulation, sections 1 and 2 of this article will describe heat transfer within the body, heat exchange between the body and the environment, and the physiological mechanisms that promote heat loss.

1. Heat Exchange

During exercise, the release of energy as heat and the concomitant rise in body core temperature evoke powerful physiological mechanisms to promote heat loss. However, for heat loss to occur, excess heat should first be transported from the core to the skin where heat can be lost to the environment.[1] As a result of its relatively low metabolic rate and constant blood flow, the temperature of inactive skeletal muscle tends to be between 33°C and 35°C. Consequently, at rest, heat is actually transferred from core to skeletal muscle tissue. The latter changes dramatically with the onset of exercise when the increase in heat production causes the muscle temperature to rise, leading to a reversal of the temperature gradient between muscle and arterial blood. Heat is now transferred down this gradient from muscle to blood and subsequently to the body core. The rate of heat transfer from core to skin is, in turn, determined by the temperature gradient between these two and by the overall skin conductance. The latter is the sum of a fixed conductance by passive conduction across the subcutaneous fat and a variable conductance by convective heat transfer to the cutaneous circulation.[2] The ability to modulate skin blood flow, therefore, constitutes a powerful defence mechanism against hyperthermia and will be discussed in more detail in section 2.2. Once the metabolic heat is transferred to the skin, there are various ways in which it can be lost to the surrounding environment, including radiation, conduction, convection and evaporation. The different mechanisms by which heat can be gained or lost are defined by the heat balance equation: metabolism ± radiation ± conduction ± convection – evaporation = heat storage.[3]

Radiation is the loss or gain of heat in the form of infrared heat rays. All objects that are not at absolute zero temperature emit such rays. The human body simultaneously emits and receives radiant heat. When the body temperature is higher than that of its surroundings, a greater quantity of heat radiates from the body than to it. When a nude person at rest is placed in a thermal comfortable room, radiation accounts for approximately 60% of total heat loss.[4] The transfer of heat from a body to an object (or vice versa) or within an organism down a thermal gradient is called conduction. At a comfortable room temperature, only 3% of total body heat loss occurs by this mechanism. Heat transfer via moving gas or liquid is called convection. A small amount of convection almost always occurs because of the tendency of the air surrounding the skin to rise as it becomes heated. Consequently, an individual who is placed in a thermal comfortable room without considerable air movement still loses about 15% of his or her heat by convection to the air.[1,4] Finally, evaporative heat loss occurs by means of insensible water loss and sweating. Insensible water loss com-

prises the loss of water through ventilation and diffusion, and there are no control mechanisms that govern the rate of insensible water loss for the purpose of temperature regulation. The loss of heat by the evaporation of sweat, on the other hand, can be controlled by regulating the rate of sweating, and sweating rates of up to 3.5 L/hour have been reported in trained athletes. For every mL of water that evaporates from the body surface, 2.43kJ of heat is lost. At rest in a comfortable environment, about 25% of heat loss is due to evaporation. Of course, these percentages change with the onset of exercise, especially when the ambient temperature approaches or is higher than an individual's core temperature.[4,5]

2. Physiology of Temperature Regulation

2.1 The Role of the Hypothalamus

When the metabolic heat is eventually transported to the skin, heat loss is greatly accelerated by cutaneous vasodilation and sweating. Several lesion and stimulation studies on the brain have identified the hypothalamus as the neural structure with the highest level of thermoregulatory integration. Investigators recorded a large number of heat-sensitive neurons and about one-third as many cold-sensitive neurons in the preoptic and anterior nuclei of the hypothalamus.[6] These thermosensitive neurons effectively monitor the temperature of the blood flowing to the brain and can thus detect changes in core temperature. In addition to sensing changes in core temperature, the preoptic-anterior hypothalamus also receives afferent sensory input from thermoreceptors throughout the body, including the spinal cord, abdominal viscera, the greater veins and the skin. In this way, the thermosensitive neurons in the hypothalamus compare and integrate the central and peripheral temperature information. As a result, the hypothalamus is able to initiate the thermoregulatory response most appropriate for any given thermal stress.[7,8] The critical threshold temperature in the hypothalamus above or below which processes are initiated to increase heat production or heat loss is ≈37°C and all the temperature control mechanisms tend to bring the body temperature back to this 'set-point'.[9] This regulated level of core temperature varies ≈1°C as a result of the circadian rhythm of body temperature and the influence of the menstrual cycle and body temperature distribution.[10]

2.2 Cutaneous Vasodilation

The ability of the human body to adjust the cutaneous vasomotor tone provides an effective means of modulating skin blood flow (SkBF) and therefore heat flux from core to skin. As such, the cutaneous circulation can act as a major effector of the thermoregulatory response, but is also affected by non-thermoregulatory responses, such as the baroreflex and adjustments to dynamic exercise. During exercise, vasomotor reflexes have the effect of redistributing blood flow away from inactive tissue so that additional blood flow is provided to meet the increased metabolic demands of skeletal muscle. When thermoregulatory and non-thermoregulatory responses occur simultaneously, as they do during exercise, the cutaneous circulation is subjected to conflicting demands.[11] This is illustrated by the fact that, although the net response of SkBF to exercise is eventually one of vasodilation, there is noticeable constriction of the cutaneous blood vessels even in the presence of significant hyperthermia with the onset of exercise. Furthermore, the elevation of SkBF that accompanies a rise in body temperature during rest is delayed until a higher body temperature is reached during exercise. Finally, exercise places a limit on the capacity of the skin to dilate and when the internal temperature approaches 38°C, the increase in SkBF is attenuated.[12,13] From these observations, it can be concluded that the magnitude of SkBF during exercise is determined by competing vasoconstrictor and vasodilator influences.

The cutaneous circulation is controlled by two branches of the sympathetic nervous system: a noradrenergic active vasoconstrictor system and an active vasodilator system of uncertain neurotransmitter.[12] Experiments performed by Kellogg et al.[14] indicate that the release of an unknown co-

transmitter from cholinergic nerves is the primary mechanism of cutaneous active vasodilation. Although the precise mechanisms remain to be elucidated, it is clear that this system plays a vital role in the control of SkBF as its activation can fully dilate cutaneous arterioles, increasing whole-body SkBF to levels approaching 8 L/min, or 60% of cardiac output during heat stress.[12] The fact that SkBF is controlled by two different branches of the sympathetic nervous system raises the question of which pathway(s) actually sets the limitations of cutaneous vasodilation. In an attempt to separate the vasoconstrictor and vasodilator control of the cutaneous vasculature, a series of experiments was conducted that used a combination of laser-Doppler flowmetry and the local iontophoresis of the anti-adrenergic agent bretylium tosylate.[11,13,15] This technique allows the selective blockade of the active vasoconstrictor system in a small area of skin without affecting the active vasodilator system. The observation that the reductions in cutaneous vascular conductance that are normally seen with the onset of exercise were completely abolished at the bretylium-treated sites led to the conclusion that the cutaneous vasoconstriction that accompanies the onset of exercise is solely mediated by an increased active vasoconstrictor tone.[15] On the other hand, the exercise-induced elevation in the internal temperature threshold for cutaneous vasodilation appears to be the result of a delayed vasodilator response because the shifts in temperature threshold were identical both with and without vasoconstrictor system blockade.[11] The third non-thermoregulatory effect of exercise on SkBF, the attenuation of the rate of rise in SkBF during the later stages of prolonged dynamic exercise, is due to a similar attenuation of active vasodilator activity.[13]

As mentioned earlier in this section, besides being an efferent arm of the thermoregulatory response, SkBF is also known to respond to non-thermoregulatory responses, including baroreflexes. This baroreceptor-mediated control of SkBF can be important in the maintenance of blood pressure, particularly in hyperthermia, when the skin represents a significant fraction of the total vascular conductance. To test the hypothesis that baroreceptor unloading during dynamic exercise limits cutaneous vasodilation, several investigators had subjects perform dynamic exercise with and without baroreceptor unloading induced by lower body negative pressure (LBNP).[16-19] During exercise with LBNP, the body core temperature required to elicit cutaneous vasodilation was significantly elevated, indicating that SkBF responses during exercise can be modulated by baroreceptor unloading. The finding that the limitation in cutaneous vasodilation was quickly reversed when LBNP was removed clearly supports the view of a neurally mediated interaction such as the baroreflex instead of a time-dependent factor, such as a circulating vasoactive hormone.[19]

2.3 Sweating

Activation of eccrine sweat glands causes sweat to be secreted onto the skin surface, thereby promoting heat loss by evaporation of the water content of the sweat. These eccrine glands cover most of the body and are innervated by sympathetic cholinergic nerve fibres. Cholinergic stimulation of the sweat gland elicits the secretion of a so-called precursor fluid, the composition of which resembles that of plasma, except that it does not contain the plasma proteins. Therefore, sodium chloride is the primary electrolyte in sweat, with potassium, calcium and magnesium present in smaller amounts. As the precursor fluid runs through the duct portion of the sweat gland, its composition is modified by active reabsorption of the sodium and chloride.[1,4] As a result, sweat is hypotonic compared with plasma, and sweat sodium concentration typically ranges from 10 to 70 mmol/L, whereas that of chloride ranges from 5 to 60 mmol/L, depending on diet, sweat rate and degree of heat acclimatisation.[20] Athletes performing high-intensity exercise in the heat commonly have sweat rates of 1.0–2.5 L/hour, but sweat rates of >2.5 L/hour are not unusual when the ambient temperature is high.[21]

3. Factors Influencing Thermoregulation

3.1 The Environment

Heat loss by radiation and convection depends on the maintenance of a large temperature gradient between the skin and the surrounding air. When the temperature of the air exceeds 36°C, the gradient for heat exchange is reversed and the body now gains heat by radiation and convection instead of losing it. Heat loss by evaporation of sweat then becomes the primary means of heat dissipation. As sweat can only be effective for cooling if it evaporates, the potential for evaporative heat loss is determined by the water vapour pressure gradient between the skin and the environment and the movement of air over the skin. This means that when the relative humidity in the air is low (dry air), sweat will evaporate relatively fast. On the other hand, if the relative humidity of the surrounding air is high, the evaporation of sweat will be hindered and sweat will accumulate with little loss of body heat.[3] Therefore, exercise in a hot, humid environment may require levels of evaporative heat loss that exceed the capacity of the environment to accommodate more water vapour, resulting in a critical heat load that limits further exercise and increases the risk of developing heat illness.[22]

3.2 Dehydration

When sweating becomes the primary means of heat dissipation, sweat loss must be matched by fluid consumption to avoid dehydration. This is often difficult because the stimulus to drink is not initiated until an individual has incurred a water deficit of approximately 2% of body mass.[21] Consequently, when water intake during exercise in the heat is *ad libitum*, some dehydration is likely to occur. The physical work capacity for aerobic exercise is reduced when a person is dehydrated by marginal (1–2% total body water [TBW]) water deficits. Dehydration resulted in much larger decrements in physical work capacity in hot than in temperate environments, and it appears that the thermoregulatory system plays an important role in the reduced exercise performance mediated by a body water deficit. It has been shown that dehydration reduces both skin blood flow and sweating responses during exercise.[21,23] Because sweat is hypotonic compared with plasma, thermal dehydration results in a hyperosmotic, hypovolaemic condition. Therefore, both the singular and combined effects of plasma hyperosmolality and hypovolaemia have been proposed to mediate the reduced heat loss effector responses.[23] Although it has been shown that both conditions can adversely affect thermoregulation, core temperature responses are usually more strongly correlated with tonicity than with blood volume.[24] Several investigators have reported that hyperosmolality elevates core temperature by increasing the threshold temperature for both the initiation of skin vasodilation and sweating without affecting sensitivity (response per unit change in body temperature).[23,25,26] These shifts in threshold temperature are often interpreted as indicative of a CNS change in the thermoregulatory effector signal. Studies on single neurons in hypothalamic slice preparations suggest that osmotic effects on thermoregulation could be mediated by a direct influence of extracellular fluid osmolality on thermosensitive cells as almost half of the thermosensitive neurons decrease their firing rates in a hypertonic medium.[27,28] Besides affecting the CNS, tonicity could also exert a peripheral effect via a high interstitial osmotic pressure, which inhibits fluid availability to the sweat gland.[24] When, during exercise, an athlete's core temperature is raised as a result of hot and humid weather conditions or (severe) dehydration, there is an increased risk of developing heat illness.

4. Heat Illness

In the literature, three exercise-related heat illnesses are described: heat cramps, heat exhaustion and heat stroke. The term 'heat cramps' stems from the original description of the condition by Talbot,[29] who reported muscle cramping in persons performing physical work in hot, humid environments. However, these exercise-associated muscle cramps (EAMC) do not seem to be directly related to increases in core temperature and the observation

that heat stroke is not characterised by EAMC clearly supports the lack of a direct association between heat and EAMC.[30,31] Heat exhaustion is often described as a condition in which an athlete collapses during or after exercising in the heat. This condition is generally believed to result from dehydration-induced heat retention and is often considered to be a mild form of heat stroke that will progress to full-blown heat stroke if left untreated. However, there are a number of findings that argue against such a belief. First, in most individuals experiencing heat exhaustion, rectal temperatures are not abnormally elevated. Furthermore, there is no published evidence to support the notion that persons with heat exhaustion will develop heat stroke if left untreated or that they are more dehydrated than participants in the same events who do not develop the condition.[31] Another critical finding in a study performed by Holtzhausen et al.[32] was that 85% of all the studied runners collapsed after finishing the race; it is difficult to see why dehydration that is not severe enough to cause collapse during exercise (when the burden on the cardiovascular system is the greatest) should be a critical factor in the period after exercise. Therefore, it seems that the terms 'heat exhaustion' and 'heat syncope' are only used to describe a condition of collapse in individuals exercising in the heat and should not be misinterpreted to indicate that the collapse is caused by an elevated core temperature and is, therefore, a mild form of heat stroke.

The one condition that is unquestionably a direct result of an elevated core temperature is heat stroke. Heat stroke is a life-threatening illness that is clinically defined as a body core temperature that rises above 40°C, accompanied by a hot, dry skin and CNS abnormalities such as delirium, convulsions or coma.[33] During exercise, blood flow is redistributed to the skin for the dissipation of heat and this, together with the loss of fluids due to sweating, leads to a decline in central blood volume.[34] As mentioned in section 2.2, a reduction in central blood volume can result in the unloading of baroreceptors, which ultimately leads to a decline in sweating and SkBF. The latter can result in a rise in core temperature, which makes an individual susceptible to heat stroke. Furthermore, when splanchnic blood flow is reduced as part of the normal redistribution of cardiac output, the combined effects of splanchnic ischaemia and increased metabolic demands produce significant cellular hypoxia in the intestine and liver.[35] It has been suggested that the splanchnic hypoxia that accompanies heat stress results in the production of highly reactive oxygen and nitrogen species that accelerate mucosal injury leading to intestinal hyperpermeability and leakage of endotoxins into the bloodstream.[36] These endotoxins cause excessive activation of leukocytes and endothelial cells, which release pro-inflammatory and anti-inflammatory cytokines and cell-surface adhesion molecules, respectively. Both pyrogenic cytokines and endothelium-derived factors can interfere with normal thermoregulation by increasing the hypothalamic set-point at which sweating and vasodilation are activated, thereby increasing the risk that an individual will develop heat stroke.[33]

5. Intervention Strategies for Exercise in the Heat

Over the last decade, athletes and their coaches have shown an increasing interest in intervention strategies designed to reduce the health risks and performance decrements associated with exercise in the heat. This can in part be explained by the staging of important international sporting events in hot environments. For example, the searing summer heat of Athens significantly added to the challenge for athletes throughout the 2004 Olympic Games. The timing of some events did not help either, particularly the cycling road races and the women's marathon. The latter started at 6pm when the temperature was 34°C and it had dropped only a couple of degrees 2 hours later when the leaders were nearing the finish line. Sixteen of the 82 women who started the race did not make it to the finish line, including world record holder and pre-race favourite Paula Radcliffe, and the winning time was well off an Olympic record. This clearly illustrates that keeping cool can become an important advantage for

competitors. Two strategies that have proven to be particularly effective in reducing health problems and performance decrements associated with exercise in the heat are heat acclimatisation and rehydration, and they are therefore given special attention. Some basic outlines of a number of additional strategies including whole-body precooling, hyperhydration and clothing selection are discussed in sections 5.1–5.3.

5.1 Whole-Body Precooling

A number of studies reported that subjects exercising in the heat reached the point of voluntary exhaustion at similar and consistent body core temperatures despite different starting core temperatures or rates of heat storage.[37] Therefore, it has been proposed that a critical body temperature exists that directly accelerates exhaustion. Although the underlying mechanisms are not well understood, evidence is emerging that hyperthermia directly affects the functioning of the brain through altering cerebral blood flow and metabolism and decreasing the level of central cognitive or neuromuscular drive, which may in turn decrease muscle function or alter the perception of effort.[38] Given the presumed importance of body core temperature on eliciting exhaustion, the basis of whole-body precooling is to reduce the heat content of the body by cooling the periphery before exercise, thereby increasing the margin for metabolic heat production and increasing the time to reach the critical limiting temperature when a given exercise intensity can no longer be maintained. Whole-body precooling can be achieved by a variety of methods, including cold air cooling, cold water immersion and the use of a water-cooled garment.[39] The current body of evidence suggests that precooling is able to increase capacity for prolonged exercise at various ambient temperatures. However, regardless of the method used, the practical application at present is limited because of the time required to achieve sufficient body cooling to improve exercise performance.[40]

5.2 Hyperhydration

The observation that dehydration adversely affects thermoregulation gave rise to the idea that greater than normal body water (hyperhydration) might have thermoregulatory advantages. Although many studies have examined the effects of hyperhydration on thermoregulation in the heat, a large number have had serious design problems that confound their results (e.g. the control condition represents dehydration rather than euhydration). Some investigators reported lower core temperatures and higher sweating rates during exercise after hyperhydration, while others did not.[21] Although hyperhydration was often induced by having subjects overdrink plain water, it is now thought that glycerol solutions are more effective as a hyperhydrating agent as they reduce the rate of elimination of water.[41] Lyons et al.[42] gave subjects 2L of water with and without glycerol over a 2.5-hour period prior to treadmill walking in a hot, dry environment. Compared with the water trial, glycerol ingestion showed a substantially lower core temperature (0.7°C), a reduction in urine output and a higher sweating rate (33%). Montner et al.[43] reported that glycerol hyperhydration increased exercise time-to-exhaustion in temperate climates, but found no significant thermoregulatory advantages. These results are in accordance with a study by Latzka et al.,[44] who found that glycerol hyperhydration extended endurance time (from 30 to 34 minutes) in subjects exposed to uncompensable heat stress, but that it had no beneficial effect on thermoregulation compared with maintenance of euhydration. In summary, there are some indications that hyperhydration reduces thermal strain during exercise, but data supporting this notion are not very robust.

5.3 Clothing

The type and amount of clothing worn can have a major impact on heat dissipation during exercise.[45] Clothing generally represents a layer of insulation that imposes a barrier to heat transfer from the skin surface. When clothing interferes with the evaporation of sweat from the skin, increases in skin and core temperature, as well as a reduction in cooling

efficiency are observed.[46] Considering that evaporation of sweat is the most important mechanism of heat loss during exercise in the heat, clothing that poses the least amount of resistance to evaporation may prove beneficial. In practice, this would mean relatively minimal clothing, which can range from a basic swimsuit to a short-sleeve t-shirt and mid-thigh shorts. For a more detailed description of the relationship between clothing and thermoregulation, the reader is directed to a review by Gavin.[46]

5.4 Heat Acclimatisation

It has been well established that regular exposure to hot environments results in a number of physiological adaptations that reduce the negative effects associated with exercise in the heat. These adaptations include a decreased body core temperature at rest, decreased heart rate during exercise, increased sweat rate and sweat sensitivity, decreased sodium losses in sweat and urine, and an expanded plasma volume (PV).[47] The effect of acclimatisation on PV is extremely important in terms of cardiovascular stability as it allows for a greater stroke volume and a lowering of the heart rate. There remains some debate whether the expansion of PV is selective or rather reflects a generalised expansion of the extracellular fluid volume (ECF).[48] Recently, Patterson et al.[49] tried to solve this uncertainty by simultaneously measuring PV, ECF, interstitial fluid volume (ISF) and TBW in subjects that followed an exercise-heat acclimatisation protocol. They reported a simultaneous expansion of the resting PV, ECF, ISF and TBW following either short- or long-term heat acclimatisation and concluded that the increase in PV is part of a ubiquitous expansion of the entire ECF. The mechanism for the expansion of ECF and the subsequent increase in PV remains speculative; however, it has been suggested that the influx of protein from the cutaneous interstitial space to the vascular compartments causes an elevated colloid-osmotic pressure that will draw fluid into the intravascular compartment, thereby inducing a selective PV expansion at the expense of the ISF.[48] This notion is in sharp contrast with the findings by Patterson et al.[49] who showed that the ISF and PV exhibited similar relative expansions and that a generalised ECF expansion was the primary mechanism for the PV enlargement. Therefore, it is proposed that electrolyte retention, rather than the influx of protein, mediates the early PV expansion. There is indirect evidence from the literature to support this view, for example, the observation that acclimatisation-induced PV expansions are reduced in subjects who are fed a low-salt diet compared with subjects receiving a moderate- or high-salt diet.[50] A close relationship has also been observed between dietary sodium intake and the PV expansion that occurred in response to 3 days of endurance cycling.[51] Furthermore, when spironolactone, an aldosterone inhibitor, was administered to subjects during endurance training, the exercise-induced PV expansion was also restricted.[52] Therefore, it could well be that elevated plasma aldosterone levels, possibly in combination with other fluid-regulating hormones, result in an increased sodium and water retention by the kidney after heat acclimatisation.[53] Moreover, it is well established that heat acclimatisation improves the reabsorption of sodium from sweat, resulting in a greater amount of solute remaining in the plasma. This will result in a fluid shift from the intra- to the extracellular compartment, thereby causing a better maintenance of PV.[20] Another consistent finding after heat acclimatisation is the lowering of the temperature thresholds for both sweating and cutaneous vasodilation without the occurrence of a significant change in the slope of the relations. It is postulated that regular exposure to high temperatures results in a lowering of the set-points in the hypothalamus at which sweating and vasodilation are initiated.[54]

5.5 Practical Recommendations for Heat Acclimatisation

The process of acclimatisation to exercise in the heat begins within a few days, and full adaptation takes 7–14 days for most individuals. It is clear from table I that the systems of the human body adapt at varying rates to successive days of heat exposure. The early adaptations during heat acclimatisation primarily include an improved control of cardiovas-

Table I. Range of days required for different adaptations to occur during heat acclimatisation

Adaptation	Days of heat acclimatisation
Decrease in heart rate during exercise	3–6
Plasma volume expansion	3–6
Decrease in sweat Na^+ and Cl^- concentrations	5–10
Increase in sweat rate and sweat sensitivity	7–14
Increase in cutaneous vasodilation	7–14

cular function through an expansion of PV and a reduction in heart rate. An increase in sweat rate and cutaneous vasodilation are seen during the later stages of heat acclimatisation.[47]

Endurance-trained athletes exhibit many of the characteristics of heat-acclimatised individuals and are therefore thought to be partially adapted; however, full adaptation is not seen until at least a week is spent training in the heat.[55] It is not necessary to train every day in the heat, as it has been shown that exercising in the heat every third day for 30 days results in the same degree of acclimatisation as exercising every day for 10 days.[56] As the maintenance of an elevated body core temperature and the stimulation of sweating appear to be the critical stimuli for optimal heat acclimatisation, it has been recommended that strenuous interval training or continuous exercise should be performed at an intensity exceeding 50% of an athlete's maximal oxygen uptake.[47] There is evidence that exercise bouts of about 100 minutes are most effective for the induction of heat acclimatisation and that there is no advantage in spending longer periods exercising in the heat.[57] Unfortunately, heat acclimatisation is a transient process and will gradually disappear if not maintained by repeated exercise-heat exposure. It appears that the first physiological adaptations to occur during heat acclimatisation are also the first to be lost.[47] There is considerable variability in the results of studies concerning the rate of decay of heat acclimatisation, as some authors report significant losses of heat acclimatisation in less than a week, whereas others show that acclimatisation responses are fairly well maintained for up to a month. In general, most studies show that dry-heat acclimatisation is better retained than humid-heat acclimatisation and that high levels of aerobic fitness are also associated with a greater retention of heat acclimatisation.[55]

5.6 Rehydration

Although athletes may be tempted to believe that the need for fluid replacement will decrease as they become adjusted to the heat, heat acclimatisation will actually increase the requirement for fluid replacement because of the earlier onset of sweating.[47] In addition, core temperature responses after dehydration are the same for unacclimatised and acclimatised individuals, indicating that the advantages conferred by heat acclimatisation are abolished by dehydration.[21] Rehydration during exercise in the heat should therefore be made a clear priority. However, there is little agreement on the formulation of an optimal fluid replacement beverage. Factors that influence the effectiveness of a beverage as a fluid replacement include its rate of gastric emptying, intestinal absorption and how well the fluids are retained in the intra- and extracellular fluid compartments.[58]

The rate of gastric emptying is closely related to gastric volume. The larger the volume consumed, the greater the gastric emptying rate, up to at least 600mL. Therefore, when a high rate of gastric emptying is desirable, this can be accomplished by keeping gastric volume high by repeated drinking.[59] However, this volume effect can be overruled by the chemical composition of a drink. For example, the addition of carbohydrate to sports drinks can slow the rate of gastric emptying by its effect on energy content and osmolality, which are believed to exert their control of gastric emptying via the activity of receptors in the small intestine.[60] In an attempt to determine the separate effects of osmolality and energy content on gastric emptying, investigators used mixtures of free glucose and glucose polymers so that they could vary energy density and osmolality independently. The results of this study clearly show the greater importance of energy content in the control of gastric emptying; solutions with different energy content but similar osmolality emptied at

different rates, but where the energy content was similar, even large differences in osmolality had relatively little effect on the emptying rate.[61]

Once the gastric contents are emptied into the small intestine, fluids must be absorbed before any beneficial effects of rehydration can occur. Absorption of water is mainly a passive process caused by the creation of local osmotic gradients that promote net movement of water out of the intestinal lumen. Most sports drinks contain moderate amounts of carbohydrate, which, besides providing an energy source for the working muscles, will improve water absorption by producing favourable osmotic gradients.[62] Glucose and sodium are absorbed by a common membrane carrier in the brush border of the proximal small intestine and this carrier appears to transport two sodium molecules for each molecule of glucose. The transport of glucose and sodium into the cell via this membrane carrier mechanism and the subsequent extrusion of sodium by the Na^+-K^+ pump in the basolateral membrane produces the osmotic gradient for fluid absorption.[63]

On the basis of the role of sodium in active nutrient transport, it might be hypothesised that the addition of sodium in sports drinks will improve the absorption of water and glucose by activating a greater number of intestinal carrier proteins. However, a study by Gisolfi et al.[58] showed that the addition of sodium in concentrations of 0, 25 and 50 mmol/L in a 6% carbohydrate solution produced similar effects on the absorption of water, sodium and glucose in the duodenum. It seems that the glucose in the solution was the more important factor for enhancing intestinal water absorption compared with plain water, as glucose alone was just as effective as glucose plus sodium. An explanation for this lack of effect comes from the observation that within only 10cm of the duodenum, intestinal and perhaps pancreatic secretions produce a luminal sodium concentration of 40 mmol/L, which is twice the amount included in most sports drinks.[58] Furthermore, in addition to transcellular carrier-mediated transport, glucose, salt and water are transported by a process known as 'solution drag' through the paracellular pathway. Results from an animal model suggest that the presence of glucose in the intestinal lumen opens the tight junctions between brush border cells by activating cytoskeletal elements within the enterocyte.[64] This permits maximal uptake of fluid and solutes, especially when glucose concentrations are well above those required to saturate the membrane carrier.[65]

Although the study by Gisolfi and Duchman[59] showed that sodium up to 50 mmol/L in a 6% carbohydrate solution did not significantly alter fluid or glucose absorption, there are other considerations for the inclusion of sodium in sports drinks. First, moderate amounts of sodium will improve palatability for most individuals, which increases voluntary consumption. More importantly, the addition of sodium to a fluid replacement beverage will dramatically improve the retention of water after exercise-induced dehydration. This is especially important in events where athletes have to perform repeated bouts of exercise. The importance of the addition of sodium to rehydration beverages has been systematically evaluated by Maughan and Leiper.[66] They dehydrated subjects by the equivalent of 2% of body mass by intermittent exercise in the heat after which the subjects had to ingest a test drink with a volume equal to 150% of the fluid lost. These test drinks contained sodium 0, 25, 50 or 100 mmol/L. It was evident that urine output in the hours after exercise was inversely proportional to the sodium content of the ingested fluid. The subjects only remained in positive fluid balance when the amount of sodium in the test drink exceeded 50 mmol/L. These results were confirmed in a study by Shirreffs et al.[67] who showed that even when a volume equal to twice the amount lost in sweat is ingested, subjects could not remain in positive fluid balance when a low sodium drink (23 mmol/L) was consumed. A positive fluid balance was eventually maintained when drinks containing 61 mmol/L of sodium were consumed in amounts ≥1.5 times the loss of water.

5.7 Practical Recommendations for Rehydration

To maintain adequate hydration, it is generally recommended that athletes consume fluids at a rate that closely matches their loss of water through sweating and urine losses.[68] This generally requires the ingestion of 200–300mL of fluid every 10–20 minutes.[69] However, as it takes 20–30 minutes for ingested fluids to be distributed throughout the body after gastric emptying, intestinal absorption and osmotic flow, the beneficial effects of fluid intake during events lasting <20–30 minutes may be small.[70] Moreover, the levels of exercise-induced dehydration over this time period are often low, which limits the effects of fluid intake during moderate-term exercise. Athletes who compete in events lasting >30 minutes are advised to drink 200–300mL of their preferred sports drink just before exercise and to continue drinking the same sports drink throughout the event until there are 20 minutes remaining, after which little extra fluid is ingested. As mentioned in section 5.6, an important factor with regard to the effectiveness of a sports drink is its rate of gastric emptying and maintaining 400–600mL of fluid in the stomach will optimise gastric emptying.[68] However, drinking higher volumes of fluid can negatively affect performance because of the time lost in obtaining and drinking more fluid together with gastric discomfort that may be encountered. Therefore, from a performance point of view, athletes may allow themselves to drink less than what is needed for full rehydration and finish with up to 2% of body mass loss. However, when safety is the main concern, there is no question that the closer the rate of drinking can match the rate of dehydration, the better.[71]

Although it is clear that the addition of carbohydrate to a sports drink can improve intestinal water absorption, there clearly appears to be an upper limit to the amount of carbohydrate that can be added without limiting fluid availability. Sports drinks that contain >7% carbohydrate are associated with a delay in gastric emptying and reduced intestinal absorption.[72,73] The latter can be explained by the fact that high carbohydrate concentrations, because of their effect on osmolality, can cause an efflux of water into the proximal small intestine, which reduces the rate of net water absorption. Properly formulated sports drinks designed specifically to maximise rehydration during exercise should therefore not contain >6–7% carbohydrate. The type of carbohydrate ingested does not appear to be critical, but there is evidence that solutions with multiple forms of carbohydrate can produce a greater absorption of solute and water than solutions with only a single form of carbohydrate, because of the activation of several different solute transport mechanisms.[73,74] The addition of sodium to sports drinks is linked to the loss of this electrolyte in sweat. When large volumes of plain water or other electrolyte-free beverages are consumed, this may result in a rapid fall of plasma sodium concentration and osmolality, which will stimulate urine output and reduce the drinking stimulus.[75] Water alone is adequate for rehydration purposes when solid food is consumed, as this replaces the electrolytes lost in sweat. There are, however, many situations where intake of solid food is not possible or deliberately avoided and, in these instances, athletes are advised to consume fluids with at least 50 mmol/L sodium.[76]

Table II. Overview of principles regarding heat acclimatisation and rehydration

Heat acclimatisation
Full adaptation takes 7–14d to be completed
Heat acclimatisation is best achieved by strenuous interval training or continuous exercise at ≥50% of maximal oxygen uptake for at least 1h every 3d
Exercise bouts of about 1.5–2.0h seem most effective for the induction of heat acclimatisation
Acclimatisation responses are maintained for at least 1wk, but probably <1mo
Rehydration
Consumption of fluids should closely match the rate of water loss
It takes 20–30 min for ingested fluids to be evenly distributed throughout the body
The use of sports drinks with a 7% carbohydrate content improves intestinal water absorption
Water retention can be optimised by the ingestion of solutions containing at least 50 mmol/L sodium in a volume >1.5 times the amount of sweat lost

A commonly used and safe technique to determine the acute loss of body water is the measurement of body mass change. The loss of body mass over the course of exercise essentially equals water loss because no other body constituent is lost at such a high rate. When body mass measurements are made with an interval of >4 hours, the body mass difference should be corrected for the net utilisation of endogenous glycogen and fat stores.[77] Table II summarises some basic principles regarding heat acclimatisation and rehydration.

6. Conclusion

During exercise, large amounts of energy are liberated as heat. To prevent a continuous rise in body core temperature, physiological mechanisms such as cutaneous vasodilation and sweating are activated to promote the loss of excess heat. However, several factors such as a hot and humid environment and thermal dehydration can negatively influence thermoregulation and may even result in serious heat illness. Fortunately, athletes can minimise the risks associated with exercise in the heat through heat acclimatisation and rehydration. The complete process of heat acclimatisation takes 7–14 days for most individuals. Strenuous interval training or prolonged moderate intensity exercise in the heat seem to be most effective in promoting heat acclimatisation. With respect to rehydration, it is recommended that athletes consume fluids at a rate that closely matches sweat and urine losses. This generally requires the ingestion of 200–300mL of fluid every 10–20 minutes, starting just before exercise and continuing until 20 minutes of exercise are remaining. It has been shown that the addition of carbohydrate solutions (≤7%) to sports drinks improve intestinal water absorption by producing favourable osmotic gradients. To achieve effective rehydration after exercise, rehydration beverages should contain moderate to high amounts of sodium (≥50 mmol/L) and should be consumed in volumes ≥1.5 times the loss of sweat.

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